

FULL ESTIMATED COST	ENTRY	SESSION
	1.92	31.62
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=> s 120 and bronchitis
 L28 0 FILE MEDLINE
 L29 5 FILE EMBASE
 L30 4 FILE SCISEARCH
 L31 3 FILE CAPLUS

TOTAL FOR ALL FILES
 L32 12 L20 AND BRONCHITIS

=> s fibrosis and asthma
 L33 1489 FILE MEDLINE
 L34 1515 FILE EMBASE
 L35 1132 FILE SCISEARCH
 L36 634 FILE CAPLUS

TOTAL FOR ALL FILES
 L37 4770 FIBROSIS AND ASTHMA

=> s 120 and bronchitis
 L38 0 FILE MEDLINE
 L39 5 FILE EMBASE
 L40 4 FILE SCISEARCH
 L41 3 FILE CAPLUS

TOTAL FOR ALL FILES
 L42 12 L20 AND BRONCHITIS

=> s 120 and bronchiectasis
 L43 0 FILE MEDLINE
 L44 0 FILE EMBASE
 L45 0 FILE SCISEARCH
 L46 1 FILE CAPLUS

TOTAL FOR ALL FILES
 L47 1 L20 AND BRONCHIECTASIS

=> s 120 and asthma
 L48 2 FILE MEDLINE
 L49 17 FILE EMBASE
 L50 2 FILE SCISEARCH
 L51 6 FILE CAPLUS

TOTAL FOR ALL FILES
 L52 27 L20 AND ASTHMA

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FILE 'MEDLINE, EMBASE, SCISEARCH' ENTERED AT 11:57:20 ON 04 APR 2003
L2          0 FILE MEDLINE
L3          0 FILE EMBASE
L4          0 FILE SCISEARCH
TOTAL FOR ALL FILES
L5          0 S TIOTROPIM AND INFLAMMATION
L6          0 FILE MEDLINE
L7          0 FILE EMBASE
L8          0 FILE SCISEARCH
TOTAL FOR ALL FILES
L9          0 S TIOTROPIM AND INFLAMMAT?

FILE 'CAPLUS' ENTERED AT 11:58:04 ON 04 APR 2003
L10         0 S TIOTROPIM AND INFLAMMAT?
L11         21 S TIOTROPIUM AND INFLAMMAT?

FILE 'MEDLINE, EMBASE, SCISEARCH' ENTERED AT 11:58:22 ON 04 APR 2003
L12         6 FILE MEDLINE
L13         25 FILE EMBASE
L14         5 FILE SCISEARCH
TOTAL FOR ALL FILES
L15         36 S TIOTROPIUM AND INFLAMMAT?

FILE 'MEDLINE, EMBASE, SCISEARCH, CAPLUS' ENTERED AT 11:58:37 ON 04 APR
2003
L16         6 FILE MEDLINE
L17         25 FILE EMBASE
L18         5 FILE SCISEARCH
L19         21 FILE CAPLUS
TOTAL FOR ALL FILES
L20         57 S TIOTROPIUM AND INFLAMMAT?
L21         45 DUP REM L20 (12 DUPLICATES REMOVED)

FILE 'STNGUIDE' ENTERED AT 12:01:54 ON 04 APR 2003

=> s rhinitis and l20
L22         0 RHINITIS AND L20

=> s fibrosis and l20
L23         0 FIBROSIS AND L20

=> s fibrosis and asthma
L24         0 FIBROSIS AND ASTHMA

=> s l20 and asthma
L25         0 L20 AND ASTHMA

=> s l20 and bronchiectasis
L26         0 L20 AND BRONCHIECTASIS

=> s l20 and bronchitis
L27         0 L20 AND BRONCHITIS

=> fil medline, embase, scisearch, caplus
COST IN U.S. DOLLARS          SINCE FILE          TOTAL

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=> s fibrosis and l20

L53 0 FILE MEDLINE
L54 1 FILE EMBASE
L55 1 FILE SCISEARCH
L56 2 FILE CAPLUS

TOTAL FOR ALL FILES

L57 4 FIBROSIS AND L20

=> s rhinitis and l20

L58 0 FILE MEDLINE
L59 1 FILE EMBASE
L60 0 FILE SCISEARCH
L61 3 FILE CAPLUS

TOTAL FOR ALL FILES

L62 4 RHINITIS AND L20

=> s l42 or l47 or l52 or l57 or l62

L63 2 FILE MEDLINE
L64 19 FILE EMBASE
L65 5 FILE SCISEARCH
L66 6 FILE CAPLUS

TOTAL FOR ALL FILES

L67 32 L42 OR L47 OR L52 OR L57 OR L62

=> dup rem l67

PROCESSING COMPLETED FOR L67

L68 28 DUP REM L67 (4 DUPLICATES REMOVED)
ANSWERS '1-2' FROM FILE MEDLINE
ANSWERS '3-20' FROM FILE EMBASE
ANSWERS '21-23' FROM FILE SCISEARCH
ANSWERS '24-28' FROM FILE CAPLUS

AN 1998:824600 SCISEARCH
GA The Genuine Article (R) Number: 131PU
TI Chronic obstructive pulmonary disease: new opportunities for drug
development
AU Barnes P J (Reprint)
CS NATL HEART & LUNG INST, IMPERIAL COLL, SCH MED, DOVEHOUSE ST, LONDON SW3
6LY, ENGLAND (Reprint)
CYA ENGLAND
SO TRENDS IN PHARMACOLOGICAL SCIENCES, (OCT 1998) Vol. 19, No. 10, pp.
415-423.
Publisher: ELSEVIER SCI LTD, THE BOULEVARD, LANGFORD LANE, KIDLINGTON,
OXFORD OX5 1GB, OXON, ENGLAND.
ISSN: 0165-6147.
DT General Review; Journal
FS LIFE
LA English
REC Reference Count: 78
AB Chronic obstructive pulmonary disease (COPD) is common and there is an
increasing worldwide prevalence. There are no available treatments to
prevent the progression of airflow obstruction, but greater understanding
of the molecular and cellular mechanisms involved in COPD has identified
many new therapeutic targets, including **inflammatory** mediators,
proteases and adhesion molecules. In this review, Peter Barnes considers
potential new drugs for this neglected disease.
CC PHARMACOLOGY & PHARMACY
STP KeyWords Plus (R): HUMAN MONONUCLEAR PHAGOCYTES; HUMAN NEUTROPHIL
ELASTASE; NECROSIS-FACTOR-ALPHA; ALVEOLAR MACROPHAGES; CHRONIC-
BRONCHITIS; ALPHA-1-ANTITRYPSIN DEFICIENCY; METALLOPROTEINASE
INHIBITORS; MUCUS HYPERSECRETION; **TIOTROPIUM** BROMIDE; SMOKING
CESSATION

L68 ANSWER 22 OF 28 SCISEARCH COPYRIGHT 2003 ISI (R)
 AN 1999:934630 SCISEARCH
 GA The Genuine Article (R) Number: 260JK
 TI Novel approaches and targets for treatment of chronic obstructive
 pulmonary disease
 AU Barnes P J (Reprint)
 CS UNIV LONDON IMPERIAL COLL SCI TECHNOL & MED, NATL HEART & LUNG INST, SCH
 MED, DOVEHOUSE ST, LONDON SW3 6LY, ENGLAND (Reprint)
 CYA ENGLAND
 SO AMERICAN JOURNAL OF RESPIRATORY AND CRITICAL CARE MEDICINE, (NOV 1999)
 Vol. 160, No. 5, Supp. [S], pp. S72-S79.
 Publisher: AMER LUNG ASSOC, 1740 BROADWAY, NEW YORK, NY 10019.
 ISSN: 1073-449X.
 DT Article; Journal
 FS LIFE; CLIN
 LA English
 REC Reference Count: 54
 AB There is a driving need to develop new and effective treatments for
 COPD. Bronchodilators are now the mainstay of symptomatic therapy and a
 new long-acting anticholinergic bronchodilator, **tiotropium**
 bromide, is now in advanced clinical trials as a once daily dry powder
 inhaler. Several **inflammatory** mediators are involved in the
 chronic neutrophilic **inflammation** that typifies COPD, including
 leukotriene B-4 and interleukin 8; for which specific receptor antagonists
 have been developed. Since the **inflammatory** process in COPD is
 essentially steroid resistant, new antiinflammatory treatments are needed.
 Drugs that may be effective include phosphodiesterase 4 inhibitors,
 NF-kappa B inhibitors, and interleukin 10. Inhibition of proteases is
 another approach and inhibitors of neutrophil elastase, cathepsins, and
 matrix metalloproteases are now in clinical development. Supply of
 endogenous antiproteases, such as alpha(1)-antitrypsin and secretory
 leukocyte protease inhibitors as recombinant proteins or by gene transfer,
 is also being explored. In future drugs that may stimulate alveolar repair
 might be developed, including retinoid receptor agonists and hepatic
 growth factor. Future directions will include earlier detection of
 disease, gene profiling to identify which smokers are at risk of COPD, and
 the development of noninvasive surrogate markers to monitor disease
 activity in order to monitor new therapies. Identification of genes that
 confer a risk for COPD in smokers may identify novel targets for drug
 development.
 CC EMERGENCY MEDICINE & CRITICAL CARE; RESPIRATORY SYSTEM
 STP KeyWords Plus (R): HUMAN NEUTROPHIL ELASTASE; AIRWAY EPITHELIAL-CELLS;
 ALVEOLAR MACROPHAGES; **TIOTROPIUM** BROMIDE; CONTROLLED TRIAL;
 CYSTIC-FIBROSIS; KAPPA-B; INHIBITOR; **ASTHMA**; COPD
 RE